# ARIZONA HOUSE OF REPRESENTATIVES Fifty-second Legislature – First Regular Session

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1-8-16

# HOUSE AD HOC COMMITTEE ON PEDIATRIC AUTOIMMUNE NEUROPSYCHIATRIC DISORDER

Report of Interim Meeting Thursday, January 7, 2016 House Hearing Room 1 -- 10:00 a.m.

Convened 10:11 a.m.

Recessed

Reconvened

Adjourned 11:13 a.m.

Members Present

Representative Heather Carter, Chair

Representative Randall Friese

Shelly Avila\*

Tess Burleson

Dr. Michael Daines

Kate Fleck

Kari Kling

Lee Miller

Dr. Suraj A. Muley

Molly Ochoa

Dr. Sydney Rice

Dr. Mark Ross

Paul Ryan

Dan Twibell

**Members Absent** 

Representative Vince Leach

Dr. Melanie Alarcio

Charles Bassett

Dr. Cari Christ

Dr. Jay Cook

Dr. Fayez Ghishan

Dr. Susan Swedo

#### **Agenda**

Attachment 1

### Request to Speak

Mr. Paul Ryan

None

<u>Name</u>

**Organization** 

Citizen

**Attachments** (Handouts)

2

Sandy Kelley Chairman Assistant

January 8, 2016

(Original attachments on file in the Office of the Chief Clerk; video archives available at http://www.azleg.gov)

<sup>\*</sup>With approval from the Speaker, Lisa Betts attended as a designee for Shelly Avila.

# ARIZONA HOUSE OF REPRESENTATIVES

# INTERIM MEETING NOTICE **OPEN TO THE PUBLIC**

## HOUSE AD HOC COMMITTEE ON PEDIATRIC AUTOIMMUNE NEUROPSYCHIATRIC DISORDER

Date:

Thursday, January 7, 2016

Time:

10:00 A.M.

Place:

HHR 3

#### **AGENDA**

1. Call to Order

2. **Opening Remarks** 

- 3. Review of Subcommittee Findings
  - Standard of Care
  - Center of Excellence
  - Child Safety
- 4. **Public Testimony**
- **Closing Comments** 5.
- 6 Adjourn

#### Members:

Representative Heather Carter, Chair

Representative Randall Friese Representative Vince Leach

Dr. Melanie Alarcio

Shelly Avila **Charles Bassett** Tess Burleson Dr. Cara Christ Dr. Jay Cook

Dr. Michael Daines

Kate Fleck

Dr. Fayez Ghishan

Kari Kling Lee Miller

Dr. Suraj A. Muley Molly Ochoa

Dr. Sydney Rice Dr. Mark Ross

Paul Rvan

Dr. Susan Swedo

Dan Twibell

1/5/16

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Dec 15, 2015 02:08 PM EST

# Strep A: Recurrent Infections Affect The Brain

A group of researchers from Columbia University Medical Center and the University of Minnesota have discovered how recurrent infections of Strep A in children can trigger immune cells to enter the brain, which causes inflammation that can lead to autoimmune neuropsychiatric disorders.

The study was done on mice, finding that immune cells travel along odor-sensing neurons that emerge from the nasal cavity, instead of breaching the blood-brain barrier directly. The findings could help develop improved diagnostic, monitoring and treatment methods for these disorders.

Group A Strep, also known as Strep A or "Streptococcus Agalactiae," is a bacterium that causes many different infections, most notably strep throat and impetigo. Recurrent infections of strep have been linked to several autoimmune neuropsychiatric disorders, including PANDAS - Pediatric Autoimmune Neuropsychiatric Disorders associated with Streptococcal infections - which can happen overnight and immediately, and include OCD and other Tourettes-like "tic" disorders, according to the Mayo Clinic.

When a person gets Strep A, the immune system ends up producing autoanitobodies that attack the body's own tissues. Strep A's bacterial cell wall confuses the immune system because it contains mimetic molecules that are similar enough to those in the human heart, kidneys, and brain tissue, according to Dritan Agalliu, an assistant professor in the department of Pathology and Cell Biology at CUMC.

Scientists were previously unaware about how these autoantibodies gained access to the brain, since normally brain vessels form the tightly bound blood-brain barrier that does not allow free movement of molecules, antibodies, or immune cells into the brain from the blood.

However, recurrent Strep A infections can trigger the body to produce Th17 cells, an immune cell that's somewhat of a Helper T Cell, in the nasal cavity. The researchers found that in Strep A-infected mice, these bacterial-specific Th17 cells move on the surface of axons from the olfactory system into the olfactory bulb of the brain, the portion that processes odor information.

"Once the Th17 cells enter the brain, they break down the blood-brain barrier, allowing autoantibodies and other Th17 cells to enter the brain and promote neuroinflammation," Agalliu said in a press release. "What's interesting is that we see abundant Group A Strep bacteria in the nose, but they never penetrate the brain. This is different from Group B Strep - the cause of bacterial meningitis - which causes neuroinflammation by entering the brain directly."

This discovery is a huge step for developing a test for definitively diagnosing PANDAS, which can currently only be diagnosed based on clinical symptoms and the presence of Strep A, or autoantibodies against brain proteins.

"We are also interested in exploring ways to treat the disorder by repairing the blood-brain barrier itself to prevent the entry of autoantibodies into the brain," Agalliu said.

The findings were published in the Journal of Clinical Investigation.

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